

and intravenous enoxaparin (30 mg) followed by angiography within 3-12 hs of randomisation and adequate revascularisation (culprit artery plus non-culprit severe diseased arteries threatening functionally important areas) with stenting or surgery. When T failed (TIMI flow grade 0 or 1) abciximab was given before stenting. Heparin was stopped in both groups after PCI. The co-primary endpoints were: infarct size (CK-MB mass, cTnT release), left ventricle 6-week angiographic evolution (volume, ejection fraction, wall motion index) and myocardial reperfusion (normalization of ST at 3 and 6 hs). The incidence of cardiac and non-cardiac events was also compared.

Results: There were no differences between primary PCI and facilitated strategies in terms of infarct size (CK-MB mass: 4768 ± 3734 vs 4602 ± 3371 ug/Lxh, $p = 0.76$; cTnT: 275 ± 211 vs 242 ± 156 ug/Lxh, $p = 0.55$), left ventricular ejection fraction at 6 weeks (56 ± 13 vs $56 \pm 12\%$, $p = 0.9$); wall motion index at six weeks (-1.1 ± 0.6 vs -1.2 ± 0.4 , $p = 0.1$) and myocardial perfusion (normalization of ST at 3 hs: 47 vs 46% , $p = 0.83$). However, in facilitated pts the incidence of complete ST resolution at 6 hs was higher (43 vs 61% , $p = 0.03$), and the rate of cardiac events at six month tends to be lower (death, reinfarction or revascularization: 14% vs 9%).

Conclusions: According to the results of the GRACIA-2 trial, the strategy of performing facilitated intervention is as safe as optimal primary PCI for pts with STEAMI. Results also suggest that both strategies have equivalent efficacy in restoring myocardial perfusion, preserving left ventricular size and function and benefiting clinical outcome.

1118-79

Drag Reduction by Polymer Infusion: A New Mechanism to Enhance Microcirculatory Perfusion for the Treatment of Ischemia

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Background: In the setting of severe, flow-limiting coronary artery stenosis, we have previously shown that drag-reducing polymers (DRPs), which are unique, blood-soluble macromolecules, have therapeutic potential because they further lower microvascular resistance and enhance perfusion, even after autoregulation is exhausted. In bench studies involving rigid tubes, DRPs reduce resistance by altering the hydrodynamic properties of blood flow. Whether changing blood rheology is their mechanism of action *in vivo*, or whether it also involves vasodilation is unclear. To test the hypothesis that DRPs decrease resistance and increase blood flow without causing vasodilation, we examined the microcirculation of the cremaster muscle in rats using intravital microscopy. **Methods:** Catheters were placed in the carotid artery for pressure measurement in 13 anesthetized rats. A flow probe was placed around the abdominal aorta. The cremaster muscle was exposed and mounted on a microscope. Red blood cell (RBC) velocity and vessel diameters were measured in arterioles (A), venules (V), and capillaries (C) pre- and post- intravenous DRP (polyethylene oxide, 1PPM). Vascular resistance was calculated as mean arterial pressure/aortic flow. In 5 controls, topical nifedipine was applied to the cremaster to confirm measurable vasoactivity in our model. **Results:** Arteriolar diameter increased $18 \pm 4\%$ ($p < 0.03$) after topical nifedipine in controls. DRP increased aortic flow (7.4 ± 1.0 vs. 5.0 ± 0.6 mL/min, $p < 0.002$) and decreased vascular resistance (14.2 ± 1.7 vs. 23.5 ± 2.7 mmHg-min/mL, $p < 0.01$). There was no significant change in diameter at any microvascular level in association with this resistance reduction. DRP increased RBC velocity in the A (13.0 ± 0.8 vs. 11.4 ± 0.6 mm/sec, $p < 0.04$), V (10.3 ± 0.4 vs. 8.5 ± 0.6 mm/sec, $p < 0.003$) and C (149 ± 34 vs. 103 ± 31 μ m/sec, $p < 0.02$). **Conclusions:** DRPs lower vascular resistance and increase flow without causing vasodilation. The rise in flow is accompanied by an increase in microvascular RBC velocity. Primary enhancement of microvascular perfusion through modulation of the rheological properties of blood represents a promising, novel therapeutic strategy for ischemic syndromes.

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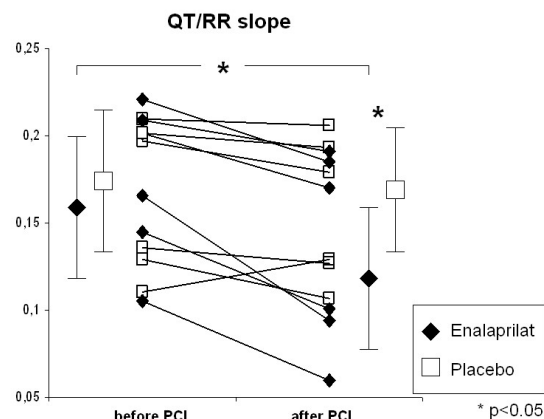
Effects of Intracoronary Low-Dose Enalaprilat on QT Dynamicity in Patients Undergoing Direct Percutaneous Coronary Intervention for Acute Myocardial Infarction

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Experimental data suggest that ACE inhibition results in an increased ventricular electrical stability in acute myocardial infarction (AMI). As electrical stability is largely dependent on ventricular repolarization, we sought to determine the impact of low-dose intracoronary application of enalaprilat (EN) as an adjunct to direct PCI on the QT/RR relationship in AMI.

Methods and Results: Twenty-two patients with AMI who underwent direct PCI (TIMI 3 flow) were randomized to EN (50 μ g) or saline (SA), given immediately after reperfusion. On hospital admission a 24-hour-Holter-ECG was initiated. Slopes of the linear QT/RR regression were determined for the time intervals before reperfusion and after reperfusion. A total of 6 patients in the EN group and 6 patients in the SA group had valid Holter-ECGs for QT analysis. Mean RR interval and QT interval were not significantly different between the EN and the SA groups before and after PCI. There were also no significant differences regarding QT/RR slopes between EN and SA groups before PCI. After PCI, QT/RR slopes significantly decreased in the EN group, whereas there were no significant alterations in the SA group.

Conclusion: Intracoronary EN therapy as an adjunct to direct PCI significantly decreases QT/RR slopes, suggesting a normalization of the coupling between heart rate and repolarization by improving electrical restitution. Thus, our findings offer new insights into possible beneficial effects of ACE inhibition on electrical stability in AMI.



1118-81

The Impact of Abciximab on Final Infarct Size and Left Ventricular Function of Patients With Acute Myocardial Infarction Undergoing Primary Stenting: Insights From the Abciximab and Carbostent Evaluation (ACE) Trial

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OBJECTIVES: We sought to evaluate the impact of abciximab, as adjunctive therapy to routine infarct-related artery (IRA) stenting, on effectiveness of myocardial reperfusion. **BACKGROUND:** The impact of abciximab on left ventricular (LV) volumes and function of patients with acute myocardial infarction (AMI) undergoing IRA stenting is not yet defined. **METHODS:** Four hundreds unselected patients with AMI were randomised 1:1 to IRA stenting alone or stenting plus abciximab. One-month gated 99m-Tc Sestamibi SPECT had to be performed in a pre-specified subgroup of 250 patients to evaluate infarct size and LV volumes. **RESULTS:** Out of the 250 patients planned for the scintigraphic assessment, 8 died within the first month, 12 dropped out of the study protocol, 13 were excluded because gated acquisition was precluded by arrhythmias or technical reasons, and 35 had a previous myocardial infarction preventing the index infarct size evaluation. Thus, 182 patients represent the final study group (99 patients were randomized to abciximab therapy and 83 to stenting alone). There were no differences in the baseline clinical and angiographic characteristics, and in discharge therapy between the 2 groups. In the abciximab group infarct size was smaller than in stenting alone group (14.3 ± 11.7 versus $19.1 \pm 13.0\%$; $p = 0.01$), resulting in smaller LV end-diastolic volume index (57.8 ± 20.0 versus 64.6 ± 20.8 mL/m²; $p = 0.03$), and LV end-systolic volume index (31.7 ± 17.4 versus 37.5 ± 18.6 mL/m²; $p = 0.03$). One-month LV ejection fraction was significantly higher in patients randomized to abciximab, as compared to stenting alone (47.4 ± 11.3 versus $43.9 \pm 11.7\%$; $p = 0.04$). Reinfarction within 30 days occurred in 3 (4%) of patients of the stenting alone group as compared to none of the abciximab group ($p = 0.056$). After excluding the 3 patients who experienced a reinfarction, the beneficial effect of abciximab on LV volumes and function still persists. **CONCLUSIONS:** The use of abciximab, as a standard adjunctive therapy to IRA, leads to the reduction in infarct size, resulting in smaller 1-month LV volumes and better LV function. The effect is not explained by the lower reinfarction rate.

1118-82

Distinct Modes of Cardiovascular Death Associated With Impaired Epicardial and Myocardial Perfusion Following Fibrinolysis for ST Elevation Myocardial Infarction

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Background: Despite fibrinolysis for ST-elevation MI (STEMI), a significant mortality remains. A comprehensive analysis of cause of death and angiographic features could provide important information for therapeutic decision-making and understanding pathophysiology.

Methods: 3683 patients from an angiographic database STEMI fibrinolysis trials were analyzed. 180 deaths (4.9%) were observed. Angiographic features were measured by a blinded core lab.

Results: Causes of death included: CHF/Shock (22%), Stroke/ICH (18%), dysrhythmia (16%), cardiac rupture (13%), MI (12%), other cardiac (7%), hemorrhage (3%). Those with cardiac death compared to survivors were less likely to have TIMI flow grade (TFG) 2-3 ($p < 0.001$) and TIMI Myocardial Perfusion Grade (TMPG) 2-3 ($p = .004$); had higher Corrected Frame Counts (CTFC, 68 vs. 52 , $p < 0.001$), and were more likely to have 3 vessel CAD ($p = 0.001$) or thrombus ($p = 0.001$). Among cardiac deaths, TFG 0-1, higher CTFC, were associated with CHF/Shock (figure); TFG 0-1 and TMPG 0-1 were associated with dysrhythmia (figure) and cardiac rupture ($p < 0.05$). There were no angiographic correlates of CVA/hemorrhage.

Conclusions: Impairment of epicardial and myocardial perfusion were associated with cardiovascular mortality. Angiographic features differed among causes of death. These angiographic correlates with mortality may aid in understanding the limitations of reperfusion therapy and the search for new modalities to reduce mortality in STEMI.